THE EFFECT OF EXERCISE ON LUNG DISTENSIBILITY AND RESPIRATORY WORK IN MITRAL STENOSIS

BY

GRAHAM W. HAYWARD AND J. M. S. KNOTT

From St. Bartholomew's Hospital

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The selection of patients with mitral stenosis for valvotomy is considerably influenced by the degree of dyspnœa of which the patient complains, and in many cases the history of progressive dyspnœa with episodes of acute pulmonary œdema or hæmoptysis makes the need for operation obvious. In some cases, however, fatigue is confused with dyspnœa as a reason for limiting physical activity or the symptoms of the effort syndrome may be superimposed so that some objective measurement of the degree of disability might be of value. Attempts to correlate the degree of dyspnœa with the circulatory changes in the heart and lungs, measured by means of the cardiac catheter, or with results of tests of respiratory efficiency have given conflicting results. Most of these studies have been carried out with the patient at rest but even when repeated during steady exercise the same lack of correlation has been reported (West *et al.*, 1953).

Pathologists have long been familiar with the fact that the lungs of patients dying in heart failure were more rigid than normal lungs, but it was not until 1934 that Christie and Meakins (1934) measured *in vivo* the distensibility of the lungs in patients with congestive heart failure by means of simultaneous tracings of the tidal air and the intrapleural pressure fluctuations. They showed that in heart failure the lungs were more rigid than normal, probably because of pulmonary congestion, and that with clinical improvement the distensibility of the lungs improved. There was thought to be a direct relationship between the degree of lung rigidity and the amount of dyspnœa and it was later suggested that cardiac dyspnœa was reflex in origin, the increased lung rigidity through the Hering-Breuer reflex causing the rapid shallow breathing of heart failure (Christie, 1938). Because of the necessity of inducing a small pneumothorax to record the changes in intrapleural pressure little use has been made of the method of Christie and Meakins (1934) of measuring the pulmonary distensibility in cardiac patients. Dornhorst and Leathart (1952) have shown that an accurate record of changes in intrathoracic pressure can be obtained by recording the pressure changes in the œsophagus and this method has been used to study on a larger scale changes in lung distensibility and respiratory work in heart failure (Marshall *et al.*, 1954).

If the degree of cardiac dyspnœa is directly related to the increased rigidity of the lungs, the measurement of pulmonary distensibility might be of help in assessing the amount of disability. In this investigation the relationship between dyspnœa and pulmonary distensibility and respiratory work has been studied in 30 patients with mitral stenosis. Measurements were made at rest, and during and immediately after exercise, and in 6 patients were repeated 3–6 months after mitral valvotomy.

METHODS

Changes in intrathoracic pressure were measured using an œsophageal catheter and these were recorded simultaneously with records of the tidal air obtained with the pneumotachograph and capacitance manometer. From these records the distensibility of the lung was calculated and expressed as the pleural pressure change in cm. of water necessary to produce 1000 ml. of tidal

volume change. This is the coefficient of elastic resistance as defined by Marshall *et al.* (1954). In addition the respiratory work was calculated according to the method of Dean and Visscher (1941) and was expressed as kilogram metres/minute. All measurements were made with the patient sitting on a bicycle ergometer. The first record was obtained with the patient at rest, but no attempt was made to have the patients in a basal condition. The patients were then exercised until they were obviously dyspnœic. No attempt was made to standardize the amount of exercise each patient took to reach this point. The patients were then instructed to stop cycling and a third record was taken during the first minute after stopping and while they were still dyspnœic.

The 30 patients with mitral stenosis who were investigated had all been sent to hospital for assessment of their suitability for mitral valvotomy. They were placed in one of the following four grades according to the degree of their dyspnœa.

Grade 1. Dyspnœa after relatively severe exertion.
Grade 2. Dyspnœa on moderate exertion.
Grade 3. Dyspnœa that was severe, with marked limitation of activities and in many cases with a history of cardiac asthma.
Grade 4. Incapacitating dyspnœa with clinical signs of pulmonary congestion and often right-sided heart failure.

Mitral stenosis was judged to be the predominant valve lesion in all cases, although in eight the murmur of aortic incompetence was present.

RESULTS

The distensibility of the lungs as expressed by the coefficient of elastic resistance measured at rest, when dyspnœic during exercise and during the first minute after exercise for the four grades

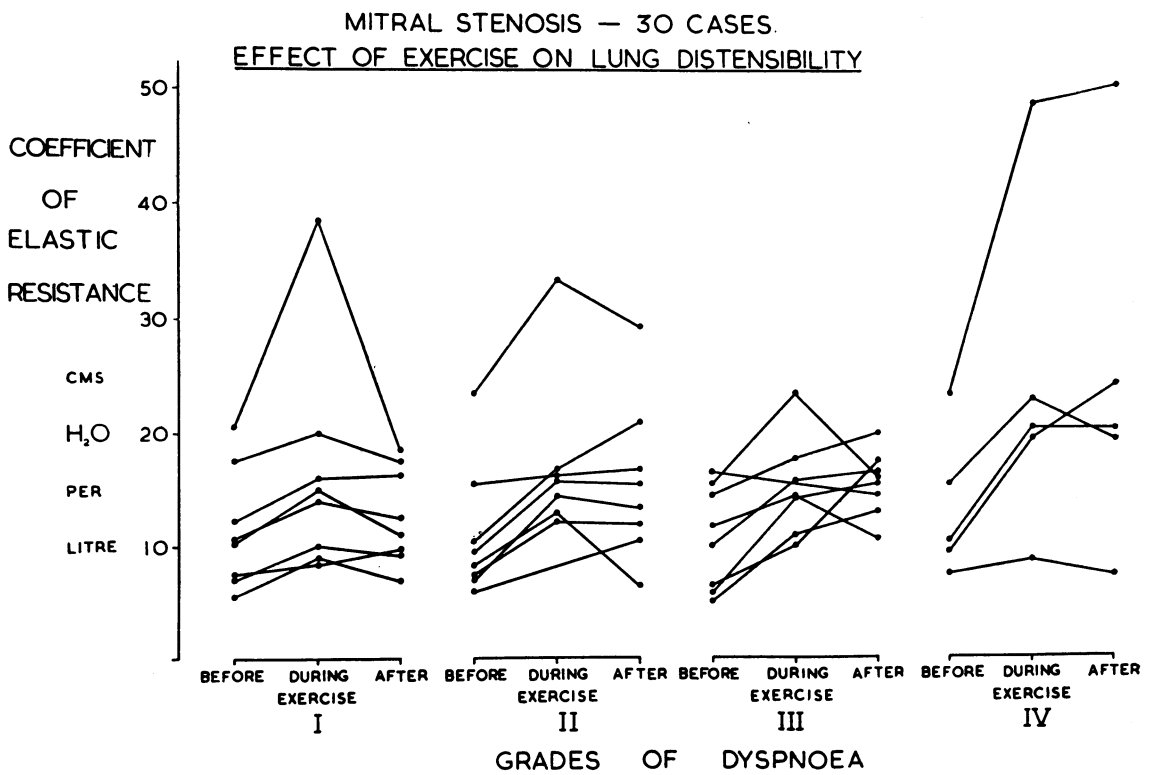

FIG. 1.—The effect of exercise on lung distensibility in 30 patients with mitral stenosis.

TABLE I

THE WORK OF BREATHING AND LUNG DISTENSIBILITY IN 30 PATIENTS WITH MITRAL STENOSIS MEASURED AT REST, DURING AND AFTER EXERCISE

Subject	Age and sex	Grade of dyspnœa	Rest		Exercise			
					During		After	
			Coefficient of elastic resistance (cm. H_2O/l)	Work in kg./m./min.	Coefficient of elastic resistance (cm. H_2O/l)	Work in kg./m./min.	Coefficient of elastic resistance (cm. H_2O/l)	Work in kg./m./min.
E. A.	F. 37	1	5·7	1·31	9·0	1·75	7·1	1·28
M. M.	F. 50	1	7·0	0·42	10·0	1·74	9·4	1·10
W. M.	F. 31	1	7·4	0·68	8·3	2·29	9·6	3·92
A. S.	M. 27	1	10·2	0·59	15·1	5·0	11·4	2·48
H. G.	F. 22	1	10·9	0·59	14·2	2·55	12·5	2·73
M. W.	F. 44	1	12·2	0·65	16·0	1·42	16·2	1·34
E. K.	F. 36	1	17·7	0·8	20·0	0·83	17·7	1·19
N. L.	F. 47	1	20·3	1·02	38·4	2·62	18·7	3·42
A. P.	F. 30	2	8·3	0·71	13·0	1·47	6·4	1·89
A. P.	F. 38	2	6·0	1·14	—	—	10·7	1·53
R. M.	M. 40	2	7·3	1·24	12·3	2·1	12·0	2·80
G. W.	M. 40	2	7·2	1·12	14·7	3·81	13·5	8·05
A. S.	F. 54	2	9·3	0·51	15·7	1·3	15·7	1·35
E. G.	F. 40	2	15·7	0·21	16·2	1·92	16·6	0·68
A. B.	F. 32	2	10·6	0·21	16·9	0·85	21·0	1·28
R. B.	F. 21	2	24·2	1·11	33·4	3·38	29·8	3·60
J. E.	M. 43	3	5·1	0·39	11·4	1·43	13·0	0·82
L. L.	F. 36	3	16·4	0·66	—	—	14·8	1·42
S. V.	F. 36	3	5·6	0·52	14·3	1·67	16·1	1·90
P. F.	F. 42	3	20·4	0·55	23·5	2·81	15·5	1·84
E. H.	F. 39	3	10·6	0·88	15·8	3·94	16·7	2·06
L. W.	F. 27	3	9·9	1·77	10·0	3·3	17·4	3·00
M. S.	F. 20	3	14·5	1·15	17·9	4·3	20·2	2·06
J. A.	F. 38	3	9·95	1·31	13·5	5·07	10·6	2·68
H. W.	M. 44	4	7·3	0·47	8·9	3·0	7·6	1·95
M. H.	F. 22	4	15·2	0·54	23·4	1·9	19·8	2·24
E. A.	M. 30	4	10·4	0·68	20·9	4·83	20·9	2·90
J. H.	F. 26	4	21·6	1·12	—	—	—	—
H. M.	F. 47	4	9·4	0·76	19·4	3·7	24·4	2·50
M. D.	F. 27	4	23·2	0·72	49·0	4·26	50·4	4·54

of dyspnœa is shown in Table I and Fig. 1. The resting distensibility figures show no significant difference in all four grades of dyspnœa. In all cases the lung became more rigid and the distensibility decreased on exercise, but the distensibility when the patient is dyspnœic was approximately the same in those who were assessed clinically as having Grade 1 or Grade 4 dyspnœa. This decreased distensibility on exercise in these patients is in sharp contrast with the increased distensibility on exercise found in normal subjects (Fig. 2). The level of distensibility recorded after exercise was in most cases lower than the level during exercise, but was approximately the same for patients in all four grades of dyspnœa.

Changes in respiratory work at rest, during and after exercise in the four different grades of dyspnœa are shown in Table I and Fig. 3. The work of breathing at rest is within the same range in all four grades (0·21–1·77 kg.m./min·; normal range 0·19–0.46 kg.m./min.) and as previously reported by Marshall *et al.* (1954) increased markedly on exercise. When the patient is dyspnœic the rate of respiratory work is approximately the same whatever the previous

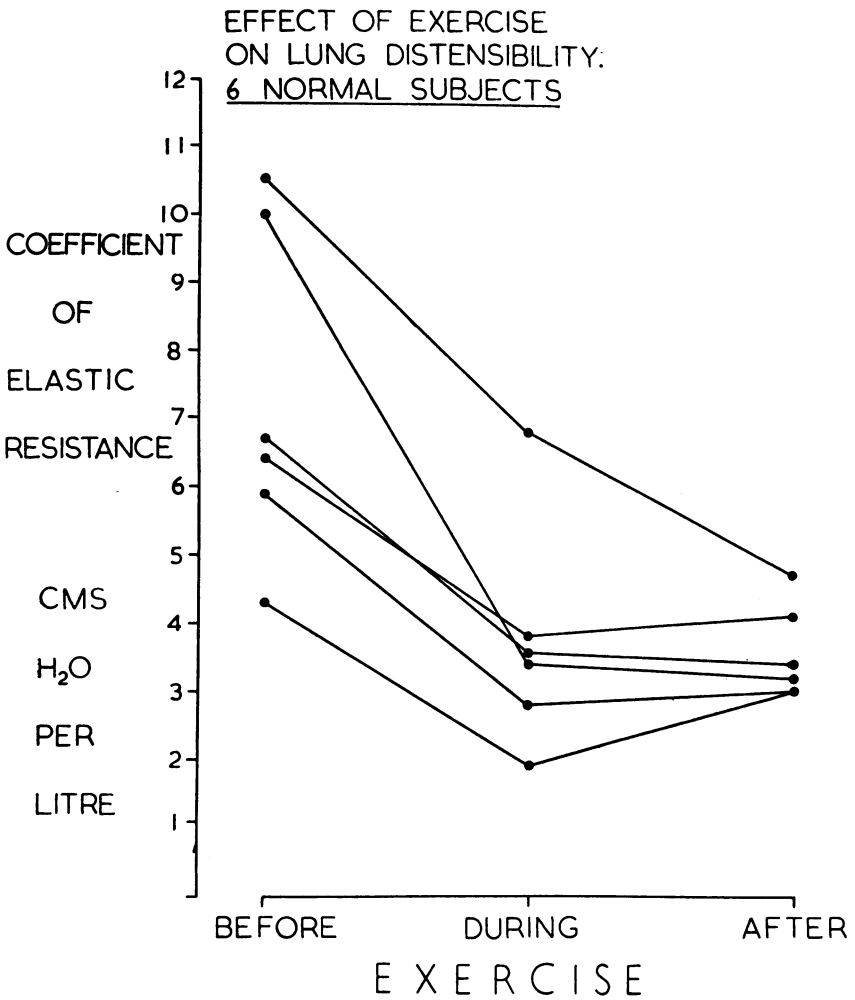

FIG. 2.—The effect of exercise on lung distensibility in 6 normal subjects.

grading of the dyspnœa. As was to be expected from their histories, the most notable difference between the patients was the duration of exercise necessary to produce dyspnœa, a very short period being necessary in Grade 4 whereas patients in Grade 1 could exercise for a much longer period. During the first minute after exercise had stopped, when the patient was often even more conscious of dyspnœa than during exercise, the level of respiratory work has often fallen abruptly.

Six patients were studied again 3–6 months after mitral valvotomy (Table II). Two of them were assessed as having Grade 4 dyspnœa, three Grade 3, and one as having Grade 2 dyspnœa before operation, and all showed a marked improvement in effort tolerance after operation. The mitral valve opening in all cases was small at operation, and in 5 cases a satisfactory increase in the size of the opening was produced. In the sixth case, the cusps could not be split and only a slight dilatation of the opening was possible.

The effects of the operation on lung distensibility are shown in Table II and Fig. 4. The resting distensibility was improved in four of the five successful cases. Before operation, in all cases the

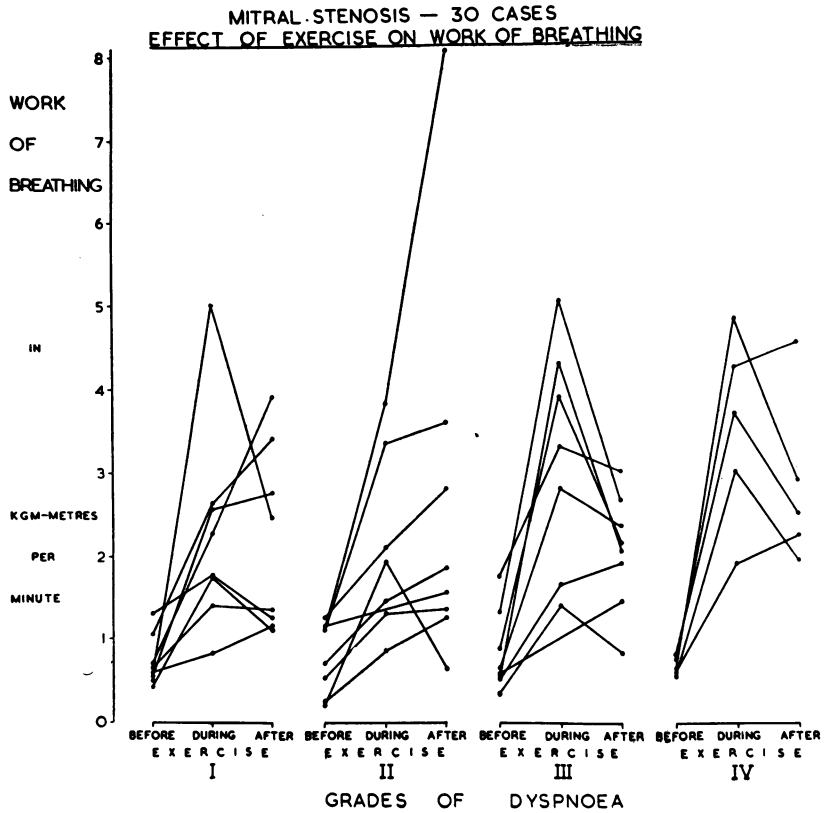

FIG. 3.—The effect of exercise on the work of breathing in 30 patients with mitral stenosis.

TABLE II

THE LUNG DISTENSIBILITY BEFORE AND AFTER MITRAL VALVOTOMY IN SIX PATIENTS

Name		Minute volume (l./min.)			Work (Kg./m./min.)		
		Rest	Exercise		Rest	Exercise	
			During	After		During	After
P. F.	Pre-op.	11·1	23·1	26·0	0·55	2·81	1·84
No. 13	Post-op.	14·9	37·1	28·7	0·57	2·18	1·79
E. H.	Pre-op.	11·8	29·6	25·6	0·88	3·94	2·06
No. 14	Post-op.	10·2	41·5	—	0·46	4·6	—
M. D.	Pre-op.	12·1	15·0	15·3	0·83	1·7	1·4
No. 18	Post-op.	9·8	19·2	21·9	0·58	1·6	2·04
A. P.	Pre-op.	15·6	11·7	23·0	0·71	1·47	1·89
No. 6	Post-op.	11·6	26·6	17·0	0·25	1·46	0·63
J. H.	Pre-op.	17·0	—	—	1·12	—	—
No. 11	Post-op.	9·2	22·5	19·8	0·36	1·72	1·60
L. W.	Pre-op.	18·3	36·4	39·3	1·77	3·3	3·0
No. 15	Post-op.	7·5	14·4	9·5	0·32	1·4	0·68

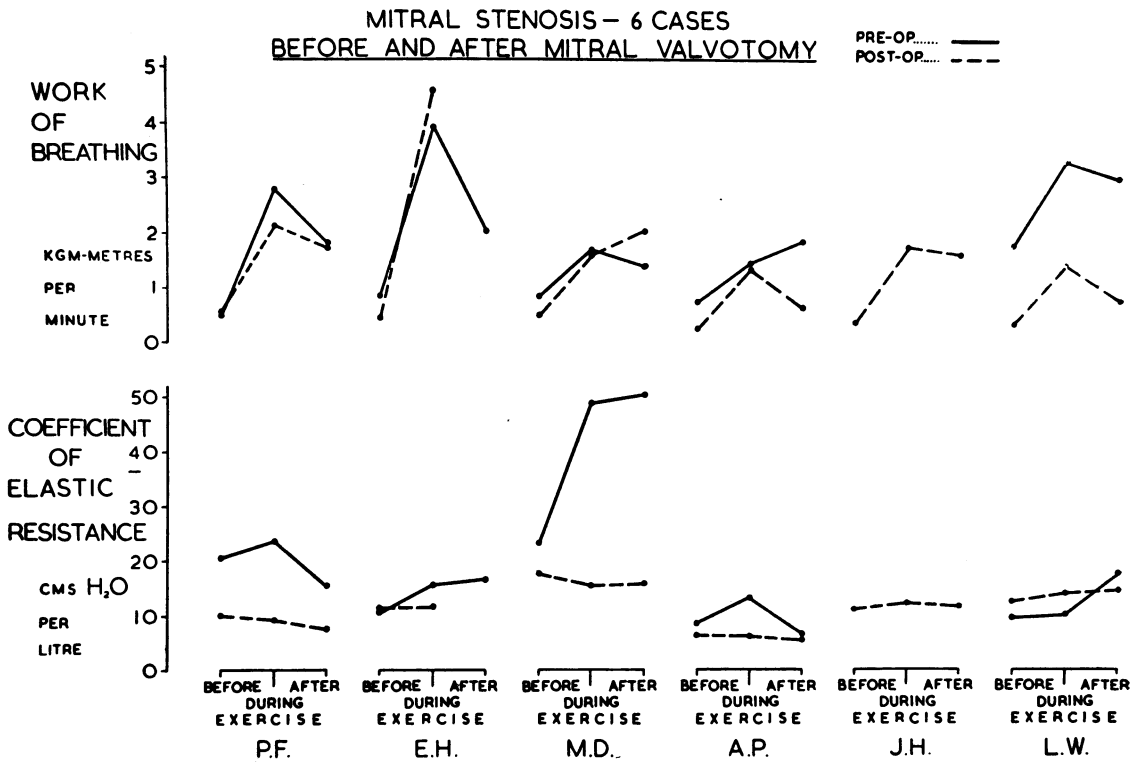

FIG. 4.—The lung distensibility and work of breathing in 6 patients before and after mitral valvotomy.

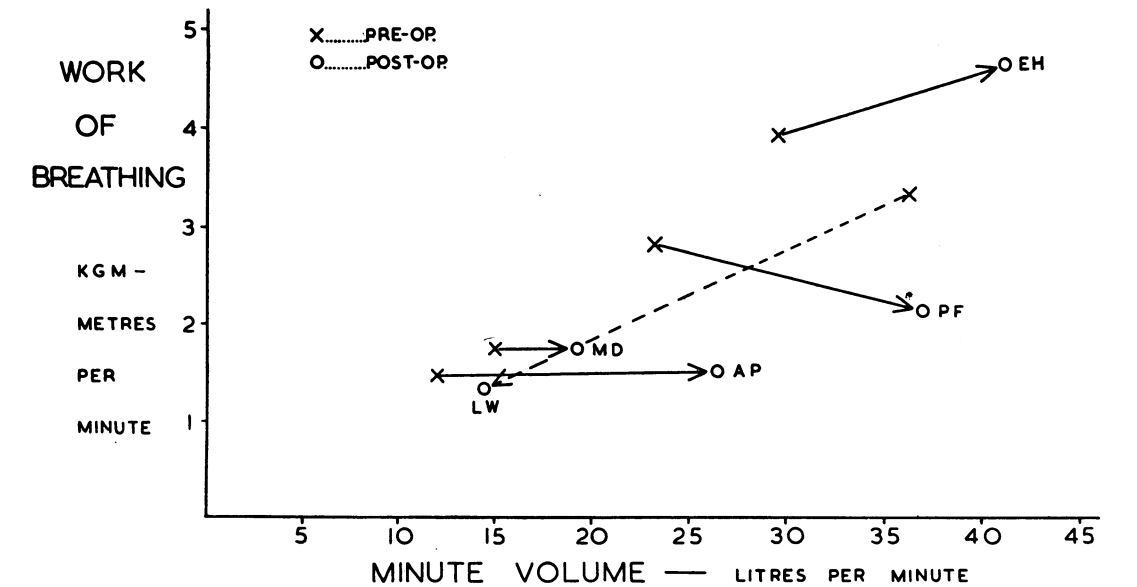

FIG. 5.—The rate of respiratory work and the minute volume when dyspnœic on exertion before and after mitral valvotomy.

lungs became more rigid on exercise and after a successful operation there was no such increase on exercise. In the sixth case (L.W.) no operation on the mitral valve was possible, and although there was marked symptomatic improvement, the lungs at rest were less distensible after the attempted valvotomy. The changes in respiratory work after operation are shown in Table III and Fig. 4. After operation the resting respiratory work was slightly lower in most cases but during exercise sufficient to cause dyspnœa, the rate of work reached the same level as before the operation. The improvement in lung distensibility is well shown by the increased minute volume of respired air after operation associated with a given amount of respiratory work (Fig. 5). In the five cases that had a satisfactory valvotomy the minute volume was increased by from 20 to 125 per cent compared with the preoperative figures, although the rate of respiratory work at the point of dyspnœa was unchanged.

TABLE III

THE WORK OF BREATHING AND MINUTE VOLUME IN SIX PATIENTS BEFORE AND AFTER MITRAL VALVOTOMY

Name, sex, age		Grade of dyspnœa	C/T ratio	Pulmonary artery mean pressures in mm./Hg		Lung distensibility (Cm. of H_2O/l. of airflow)			Operation data
						Exercise			
				Rest	Exercise	Before	During	After	
P. F. F. 42 No. 13	Pre-op. Post-op.	3 1	12·5/25 —	34 —	50 —	20·4 10·1	23·5 9·3	15·5 7·2	Valve 1·5×0·5 cm. Admits 2 fingers
E. H. F. 39 No. 14	Pre-op. Post-op.	3 1	14·3/26·5 —	50 —	73 —	10·6 11·2	15·8 11·7	16·7 —	Valve 1·0×0·5 cm. Admits 1½ fingers
M. D. F. 27 No. 18	Pre-op. Post-op.	4 0	15·0/25 —	— —	— —	23·2 17·8	49 15·3	50·4 15·8	Valve 1·0×0·5 cm. Admits 2 fingers
A. P. F. 30 No. 6	Pre-op. Post-op.	2 0	12·5/28 —	27 —	41 —	8·3 6·5	13·0 6·2	6·4 5·8	Valve 1·5×0·5 cm. Admits 1½ fingers
J. H. F. 26 No. 11	Pre-op. Post-op.	4 0	13·0/25 —	58 —	68 —	15·6 11·4	— 12·0	— 11·6	Valve 1·0×0·5 cm. Admits 1 finger
L. W. F. 27 No. 15	Pre-op. Post-op.	3 1	14·8/26 —	26	36	9·9 12·6	10·0 13·8	17·4 14·1	Valve 1·5×0·5 cm. Dilated— Not possible to split

DISCUSSION

The results reported here confirm earlier reports on the increased rigidity of the lung in mitral stenosis. The cause of the increased rigidity is generally assumed to be chronic passive congestion of the pulmonary capillary circulation although it seems probable that interstitial and alveolar œdema is of greater importance in making the lung rigid. Hayward and Knott (1954) by perfusion of the isolated animal lung have shown that although either increased pressure in the pulmonary arteries or capillary congestion cause a slight decrease in lung distensibility the effect is small compared with the changes occurring when there is œdema of the lung. Hæmodynamic studies, using the cardiac catheter, of the circulatory changes in the lungs in patients with mitral stenosis lend support to this view. The pulmonary capillary venous pressure, measured by impacting the catheter tip in a pulmonary arteriole, is normal or slightly raised at rest and during exercise rises

appreciably, frequently to a level exceeding the plasma osmotic pressure of 30 mm. Hg (Gorlin *et al.*, 1951). Clinical pulmonary œdema does not necessarily occur when the pulmonary capillary pressure exceeds the plasma osmotic pressure. The thickening of the alveolar and capillary walls and changes in the interstitial tissue (Parker and Weiss, 1936) may hinder the transudation of fluid from the capillaries, and as Drinker (1945) has shown, œdema fluid only appears in the air passages when the pulmonary lymphatics are unable to remove fluid from the interstitial tissues as fast as it escapes from the capillaries. The greatest increase in lung rigidity in the whole series occurred in the one patient in Group 4 who had obvious clinical signs of pulmonary œdema and was grossly dyspnœic. In all the other cases it is probable that the increased rigidity was due to interstitial œdema and capillary congestion, and catheter studies in these showed a pulmonary capillary pressure on exercise exceeding the plasma osmotic pressure. The effect of mitral valvotomy on the pulmonary circulation is to lower the left auricular, pulmonary capillary, and pulmonary arterial pressure, measured at rest, and to abolish or decrease the rise in pressure that occurs on exercise (Munnell and Lam, 1951). These changes are reflected in the measurements of pulmonary distensibility before and after a successful valvotomy which has relieved pulmonary congestion. The striking increase in rigidity (and decreased distensibility) which occurs on exercise before operation is abolished by a valvotomy, the lung after operation behaving as does the normal lung on exercise.

It is clear from the results of this investigation that the measurements of lung rigidity and respiratory work cannot be used as an objective measurement of dyspnœa or as a means of selecting patients for valvotomy when the degree of disability is uncertain from the history. The lack of any significant difference between the rigidity of the lungs in patients of any grade of disability who were made dyspnœic by exercise and the fact that after a successful valvotomy dyspnœa occurs with no increase in lung rigidity shows that the severity of dyspnœa cannot be directly related to the increased rigidity of the lungs in mitral stenosis.

The increased rigidity of the lung is responsible for the high rate of respiratory work which occurs on exercise and also for the low minute volume of respired air in spite of a considerable increase in the extent of the intrathoracic pressure variation on exercise. After valvotomy, when the lung does not become more rigid on exercise, the same amount of respiratory work produces a minute volume that is increased by from 20 to 125 per cent of the preoperative figures.

The rate of respiratory work increases very considerably on exercise in all these patients with mitral stenosis (0·8 kg.m./min. to 5 kg. m./min) but when dyspnœic it falls within the same range whether the patient's disability had been classified as Grade 1 or Grade 4. The chief difference between the patients was the total duration of exercise they were able to carry out before becoming dyspnœic, and the method of exercise used does not enable the total amount of work to be calculated.

Although there is no relationship between dyspnœa and the rate of respiratory work, the considerable increase that occurs on exercise is of interest as the ability to carry out muscular (and respiratory) work efficiently is closely related to the degree of disability. Studies on the mechanical efficiency of cardiac patients in carrying out muscular work show that patients with a normal or only slightly decreased effort tolerance are able to carry out light or moderate work with normal efficiency, whereas those with severe incapacity show a marked decrease in muscular efficiency (Simonson and Enzer, 1942). The decrease in efficiency runs parallel with the inability to increase the stroke volume of the heart. In the patients with severe mitral stenosis and Grade 4 disability, the circulatory adjustments that lead to efficiency in carrying out muscular work are all impaired. The cardiac output at rest is low and on exercise frequently fails to increase and may actually fall. The oxygen extraction (A-V difference) at rest is high and, as the oxygen uptake cannot be increased on exercise, the increased oxygen for the muscles can only be supplied by increasing still further the A-V oxygen difference. Lack of training, poor muscular development due to enforced inactivity, and the early onset of fatigue are other factors that decrease the efficiency of muscular performance in these patients. All these factors lead to inefficient performance of muscular work including

respiratory work, and may be partly responsible for the decreased effort tolerance which is one of the chief indications for a mitral valvotomy.

The effect of psychological factors on the onset of dyspnœa is well recognized. In the patient L.W. (Table II and Fig. 4) and in five other patients in whom it has been technically impossible to carry out a valvotomy after thoracotomy, there has been a remarkable immediate improvement in the patient's dyspnœa after operation. L.W. in her assessment after the attempted operation had improved from Grade 3 to Grade 1 despite the fact that her lungs were less distensible than before the operation. This subjective improvement was real and although it may only be temporary, illustrates the way in which psychological factors may influence effort tolerance.

SUMMARY

In mitral stenosis, the distensibility of the lungs may be decreased at rest and become further decreased on exercise as the result of pulmonary congestion and interstitial and alveolar œdema. The increase in respiratory work on exercise is many times greater than that found in the normal person. After a successful mitral valvotomy the lung behaves as does the normal lung on exercise and no decrease in distensibility occurs. The improvement in distensibility of the lung as a result of the lowering of left atrial, pulmonary capillary, and pulmonary artery pressures by mitral valvotomy is shown by the considerable increase in the minute volume of respired air which the same amount of respiratory work produces after operation. There is no relationship between the degree of dyspnœa and the rigidity of the lung, and measurements of lung distensibility when patients of all grades of disability were exercised to make them dyspnœic fell within the same range. The decreased muscular efficiency of patients with mitral stenosis may be partly responsible for their decreased effort tolerance.

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